No 1 silk, passed via the urethra in the jaws of the biopsy forceps, and the bladder filled with irrigant. The loop was then manipulated over the thermometer, pulled over the construction above the bulb, and tightened by pulling on the redundant string outside the urethra. Once the lasso was firmly attached, the bulb end of the thermometer was manipulated towards the urethra, the cystoscope was withdrawn, and the thermometer easily removed by pulling on the string.

The patient made a good postoperative recovery and was discharged next day.

Comment

The proponents of the safe period method of contraception place a lot of emphasis on the accurate daily measurement of body temperature as an aid to establishing the time of ovulation. It is suggested,[1] firstly, that the temperature should be taken immediately on waking before any activity and, secondly, that a vaginal or rectal temperature is preferable to an oral reading. It is perhaps unreasonable to expect lay people to have a detailed knowledge of anatomy, and if such manoeuvres are to be performed, clear unequivocal instructions must be issued, or the method demonstrated to the user in the family planning consultation.

We suggest that this method of extraction of cylindrical foreign bodies from the bladder is a useful adjunct to contemporary instrumentation. In this case it saved the patient considerable discomfort and a prolonged hospital stay.

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Paracetamol interference with blood glucose analysis: a potentially fatal phenomenon

Paracetamol, a popular analgesic and antipyretic, is an increasingly common cause of death from self-poisoning. Overdose with this drug has several well-recognised complications. A depressed level of consciousness in this setting should suggest one of three common complications: concomitant overdose with a sedative, hypoglycaemia, or hepatic encephalopathy.[1] We describe a case in which appropriate investigation for these complications led to treatment that nearly ended in disaster.

Case report

An unconscious 47-year-old woman with no history of diabetes was admitted with suspected paracetamol overdose. Blood analysis showed: blood glucose (by the model 23AM glucose analyser (Yellow Springs Instrument Co, Yellow Springs, OH 45487)) concentration of 54·4 mmol/l (973 mg/100 ml); paracetamol concentration of 4·5 mmol/l (680 mg/l); pH of 7·2; total CO_2 content of 11 mmol/l; and anion gap of 20 mmol/l. She was given intravenous fluids and soluble insulin at a dose of 8 units bolus and 6 units/hour infusion. Urine analysis one hour after the start of the treatment showed 0·5% glycosuria with a trace of ketonuria. Because of the discrepancy between blood glucose and urine analysis and the suspicion, based on previous data,[2] that paracetamol might be interfering substantially with glucose analysis, a sample of venous blood was checked for glucose both by YSI and Beckman glucose analysers (Beckman Instruments Inc, Fullerton, CS 92634). At the same time, a capillary sample was analysed by BM-Test-Glycemie 20-800 (Boehringer Mannheim SA, Copernico 61 Y63, Barcelona). The glucose results for the three methods were as follows: YSI 44 mmol/l (793 mg/100 ml); Beckman 4·3 mmol/l (77 mg/100 ml); BM test 2 mmol/l (36 mg/100 ml). The insulin was discontinued and an infusion of 5% dextrose and acetylcysteine started. Despite severe initial liver damage (alanine transferase activity of 5760 U/l on the third day after admission), the patient made a complete and uneventful recovery. She remained normoglycaemic for the rest of her stay in the ward. Her coma was subsequently attributed to concomitant benzodiazepine overdose.

Comment

Paracetamol interference with blood glucose analysis is not widely recognised. The manual for the YSI analyser, however, recognises paracetamol as an interfering agent, though the magnitude of this interference is at variance with our experience with this patient. According to the manual, at a paracetamol concentration of 0·46 mmol/l = 69·5 mg/l the interference is equivalent to 0·25 mmol/l (4·5 mg/100 ml) of glucose. This would predict a glucose overestimation of no more than 2·45 mmol/l (44 mg/100 ml) rather than the actual overestimation of nearly 40 mmol/l (720 mg/100 ml) in our patient. Farrance and Aldons undertook a study of the effect of paracetamol on glucose analysis by the YSI analyser and concluded that 1·0 mmol/l (151 mg/l) of paracetamol causes an apparent glucose increase of 1·4 mmol/l (25 mg/100 ml).[2] This would predict a glucose overestimation of about 6·30 mmol/l (113 mg/100 ml) in our patient. Our experience is perhaps more in accordance with that of Fleetwood and Robinson,[3] who found that the magnitude of paracetamol interference was almost threefold greater than that reported by Farrance and Aldons. The manufacturers now suggest that adding pure paracetamol to serum samples—as both the above groups did—to simulate a clinical state of affairs may exaggerate the interference, as the normal metabolic pathways of the body rapidly convert paracetamol to its conjugates, which apparently exhibit much less interference than the parent compound. This criticism obviously cannot apply to our case.

It is worth remembering that both the Beckman analyser and the BM test use a different mechanism for glucose analysis and do not therefore suffer from the disadvantage of paracetamol interference.

This report emphasises the importance of careful interpretation of hyperglycaemia in patients with paracetamol overdosage as the consequences of treatment for spurious hyperglycaemia might well be fatal.

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High concentrations of thyroid-stimulating hormone in untreated glucocorticoid deficiency: indication of primary hypothyroidism?

The association of autoimmune hypothyroidism with adrenal failure is well documented.[1] Glucocorticoid excess, however, can suppress the secretion of thyroid-stimulating hormone, and conversely its secretion may be increased in glucocorticoid deficiency in the absence of thyroid disease.[2] We have therefore examined the effect of cortisol deficiency on thyroid function tests before and after glucocorticoid replacement in patients presenting with Addison's disease or isolated adrenocorticotropic hormone deficiency.

Patients, methods, and results

Eleven consecutive cases of Addison's disease had been diagnosed by the cortisol response to synacthen stimulation and the measurement of adrenocorticotropic hormone and aldosterone concentrations and plasma renin

Thyroid function tests in patients with cortisol deficiency before and after cortisol replacement

Case No	Age at diagnosis (years)	Sex	Treatment interval (months)	Free thyroxine index		Thyroid-stimulating hormone (mU/l)	
				Before replacement	After replacement	Before replacement	After replacement
1	48	M	72	35	76	31	1·1
2	45	F	96	24	32	61·4	1·4
3	21	M	8	77	88	28	4·8
4*	60	M	12	62	123	17·6	3·8

*Isolated adrenocorticotropic hormone deficiency.

activity. Two cases of isolated adrenocorticotropic hormone deficiency had been diagnosed by standard tests of the hypothalamus-pituitary-adrenal axis. All patients had tests of thyroid function performed at presentation, which were repeated at intervals after glucocorticoid treatment.

Six of the patients with Addison's disease had normal free thyroxine indices (normal range 50-160) and thyroid-stimulating hormone concentrations (normal range 0-5 mU/l) at presentation. Five patients had raised thyroid-stimulating hormone concentrations; four of these also had low free thyroxine indices and therefore received thyroxine treatment. In two of these patients the treatment was subsequently stopped; the free thyroxine index fell rapidly in one (free thyroxine index 32 at four weeks with normal concentration of thyroid-stimulating hormone; see table, case 2), who felt unwell and was treated again with thyroxine. The other patient (see table, case 1) remained euthyroid and was well without taking thyroxine 14 weeks later.

Both patients with isolated adrenocorticotropic hormone deficiency had raised thyroid-stimulating hormone concentrations at diagnosis. One had a normal free thyroxine index and raised thyroid-stimulating hormone concentration, which became normal with cortisol replacement (case 4). The other had a low/normal free thyroxine index and raised thyroid-stimulating hormone concentration and is taking thyroxine. Details of patients with high thyroid-stimulating hormone concentrations and who were restudied without receiving thyroxine (two cases) or were restudied after thyroxine was stopped (two cases) are shown in the table.

No difference was found in the severity of cortisol deficiency in those patients with and without raised thyroid-stimulating hormone concentrations as judged by the pretreatment concentration of cortisol (at 8 00 am) (mean 270±54, range 84-580 nmol/l *v* mean 140±39, range 20-317 nmol respectively) or cortisol increment after 250 μg synacthen intramuscularly (mean 99±53, range 0-285 nmol/l *v* mean 124±50, range 0-248 nmol/l respectively).

Of the five cases of Addison's disease with raised thyroid-stimulating hormone concentrations (at diagnosis), three had positive test results for both adrenal and thyroid microsomal antibodies and two also had antithyroglobulin antibodies. Of six patients with normal thyroid-stimulating hormone concentrations, adrenal antibodies were found in three, but none had thyroid antibodies. The two cases with adrenocorticotropic hormone deficiency were both autoantibody-negative.

Comment

Seven of the 13 cases with cortisol deficiency had raised concentrations of thyroid-stimulating hormone. Of these, two had normal thyroid-stimulating hormone concentrations after glucocorticoid replacement and were euthyroid two years and five months respectively after treatment. One further patient has since been taken off thyroxine and has remained euthyroid. The two patients with Addison's disease presenting with raised thyroid-stimulating hormone concentrations and who are now euthyroid both had positive test results for adrenal and thyroid antibodies. This reversion to normal values of thyroid-stimulating hormone after cortisol replacement could be due to resolution of autoimmune thyroid disease. Indeed, steroid treatment may improve thyroid function in Hashimoto's disease but only in high dosage.[3] Alternatively, chronic cortisol deficiency may impair the thyroid response to thyroid-stimulating hormone or promote secretion of the hormone directly.[2 4 5] The changes in our patients with isolated adrenocorticotropic hormone deficiency (both of whom had no thyroid antibodies) favour this latter mechanism. Several factors may, of course, operate together, particularly in those patients with thyroid antibodies.

These cases indicate that excess thyroid-stimulating hormone is not necessarily an indicator of long-term thyroid hypofunction in untreated cases of glucorticoid deficiency. Indeed, thyroid-stimulating hormone concentrations often returned to normal with treatment. This is well illustrated by the patient who was receiving thyroxine for six years before treatment was discontinued.

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Guillain-Barre syndrome associated with Campylobacter infection

The term "acute infective polyneuritis" supposes an infective agent as its cause, though this has rarely been confirmed. A preceding gastrointestinal illness has been noted in 10-20% of cases,[1] but again an infective agent has rarely been isolated. We report a case of Guillain-Barré syndrome which occurred after a gastrointestinal illness associated with *Campylobacter* in the stools and a raised serum antibody titre to *Campylobacter*.

Case report

A 45-year-old man was admitted as an emergency with a five-day history of diarrhoea, abdominal pain, and rectal bleeding. Five days before admission he had been taking the analgesic zomepirac sodium (Zomax) for backache. On admission he was febrile with a temperature of 37·5°C; he had familial clubbing and acute tenderness both suprapubically and anteriorly on rectal examination. A barium enema showed only a few small diverticula in the sigmoid colon. He was treated initially with oral ampicillin and metronidazole. Seven days after admission a stool culture grew *Campylobacter* and his treatment was changed to oral erythromycin. Subsequently, a serum antibody titre to *Campylobacter* was 1/600. Three days after starting erythromycin he developed weakness of his left arm and an absent left triceps jerk. This rapidly progressed to symmetrical and pronounced weakness and wasting of his arms and legs, which left him with little shoulder movement, minimal finger flexion, and the ability just to flex his legs and dorsiflex his left foot. Within three days all tendon reflexes were lost. There were no cranial nerve lesions and no abnormal sensory signs apart from hyperaesthesia of the hands and feet.

The results of investigations which proved to be normal included serum electrolyte estimations, chest radiography, cytomegalovirus and mycoplasma titres, thyroid function tests, creatinine phosphokinase, serum lead, and immunoglobulin concentrations, monospot test, and urinary porphyrin concentrations. Haemoglobin was 15·4 g/dl, white cell count $14{\cdot}0\times10^9$/l with an 88% neutrophilia showing a left shift. Cerebrospinal fluid obtained 13 days after the onset of polyneuritis showed no cells but a protein concentration of 650 g/l rising to 900 g/l a week later. Repeated electrophysiological studies (from 21 days after the onset of weakness) showed evidence of a mild-to-moderate segmental demyelination in the peripheral nerves both motor and sensory (lowest nerve conduction velocity 35 ms in the